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REVIEW OF THE LITERATURE OF 1945 PERTAINING TO BRONCHOESOPHAGOLOGY.*

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A critical analysis of the literature appearing during 1945 demonstrates that two subjects dominate the discussions in the field of bronchoesophagology: tracheobronchial tuberculosis and intratracheal or bronchial penicillin therapy. These will be reviewed in detail. Other subjects such as the discussions of bronchial adenomas, resections of the esophagus, bronchogenic carcinoma and benign tumors of the esophagus are of interest, but space will not permit including them in this review.

TRACHEOBRONCHIAL TUBERCULOSIS.

The number of important articles which appeared in the literature during 1944 and 1945 indicates an increasing awareness of tracheobronchial tuberculosis. Interest in this condition is twofold: first, as a clinical entity involving the incidence, pathology, diagnosis and treatment of bronchial tuberculosis; second, the significance of the complication of tracheobronchial tuberculosis in the pathogenesis, prognosis and treatment of pulmonary tuberculosis.

Incidence: The incidence of tracheobronchial tuberculosis in patients admitted to sanitoria varies between 10 and 15 per cent; there is an increase during their stay in the sanitoria.[7] The incidence of tracheal or bronchial lesions found

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at postmortem examination of patients dying of pulmonary tuberculosis varies between 40 per cent[7] and 70 per cent.[4] The incidence of bronchoscopically confirmed tracheobronchial tuberculosis in patients in whom it is suspected varies between 30 and 60 per cent.[6,7,8,13]. Females appear to be affected more frequently than males, 70 to 85 per cent of the cases being in females,[7,8] although in one series of 279 cases there was an equal distribution.[13] The average age of the patients is greater in the males than females.[8] There is an increasing incidence of tracheobronchial tuberculosis during the course of the pulmonary tuberculosis;[4,7] however, it may occur during any stage of the disease.[5] It is interesting to note that in a series of lobectomies for tuberculosis in which there was bronchial involvement, one-third of the cases had had symptoms of tuberculosis for less than one year.[5]

Perforation of tuberculous lymph nodes into the trachea and bronchi occurred 22 times in 1,656 routine autopsies on tuberculous patients, 1.3 per cent. The incidence was definitely higher in negroes.[1] Because of the age distribution in this series, the relatively greater incidence in adults is not considered contrary to the usual concept of the condition that it occurs most frequently in children.[7]

Pathology: Tracheal and bronchial lesions generally may be divided into four groups: submucosal involvement, ulceration, hyperplasia, and the stage of healing or fibrosis. The minimal lesion consists of tubercles in the submucosa and deeper parts and around the mucous glands; it is associated with slight submucosal edema, and scattered plasma cell and lymphocytic infiltration. This process may sometimes be quite extensive before ulceration occurs. Obstruction of the airway is usually minimal. Later, small, rather superficial, single or more frequently multiple ulcerations appear. The ulcerations next become associated with granulation tissue and hyperplastic changes. Tuberculomas may be formed. All degrees of bronchial obstruction occur. Due to the reparative powers of the bronchial mucosa, the minimal lesions may heal with little or no fibrosis. The more extensive the lesion the greater the fibrosis which occurs during the healing so

that a complete stenosis may occur. These four types of lesions may all occur in different areas in the same lesion and activity may be present at one point and healing at another.[4,5,7,10,13,14]

The tuberculous process extends from the parenchymal lesion along the bronchi to the most proximal area of involvement. While the severity of the lesion may vary along its course, the involvement of greatest severity is usually that closest to the parenchymal lesion.[5] The parenchymal lesion may be either a cavity or an area of caseation, although a cavity is more frequently the lesion found at the source of the bronchial pathology.

The most frequent site of tracheobronchial tuberculosis is in the left main bronchus (approximately 50 per cent); the next most frequent site is the right main bronchus (30 per cent).[7,13] Involvement of the right main bronchus was limited, according to most reports, to the area of the right upper lobe orifice.[7] Stenosis of a fibrous type resulting from the healing process occurred most frequently in these same areas.

Four possible mechanisms are concerned with the production of tracheobronchial tuberculosis: *1.* direct surface implantation; *2.* direct extension from the parenchymal lesion along the bronchial wall; *3.* direct extension from hilar lymph nodes; and *4.* lymphogenous or hematogenous extension.[4,5] Direct extension from the parenchymal lesion or, in adults, from hilar lymph nodes is rare.[4,5] The association of tracheobronchial tuberculosis with the severer parenchymal lesions, the increasing severity of the bronchial lesion as the parenchymal lesion is approached, and the high incidence of lesions in the left main bronchus are interpreted to mean that direct implantation is the more likely mechanism.[4,13] The high incidence of involvement of the hilar lymph nodes in cases of tracheobronchial tuberculosis and the continuity of involvement from the parenchymal lesion to the most proximal point of the tracheal or bronchial lesion has been interpreted to indicate that lymphatic spread is the more likely mechanism.[5,7]

The effect of the bronchial lesion on the lung is principally

that of obstruction. It may cause an obstructive emphysema or atelectasis and retention of secretion. These latter changes may assume dominance over the parenchymal lesion. A stenosing lesion may cause a spread of the tuberculosis to other parts of the lung, secretions containing the organisms "back-firing" during cough to be carried into normal areas. Another theory of spread is that the secretions may be sprayed out of the stenosis with an "atomizer" effect, so that on the following inspiration they are carried by the air stream into normal areas.[7]

In the 22 cases of perforation of tuberculous lymph nodes into the trachea and bronchi, nine instances were found in which there were two or more perforations. In 16 of the cases the perforations occurred in cases of progressive caseous tuberculosis of the lymph glands of the thorax. The involvement of the lymph nodes was part of the primary complex in children and was part of a lymphohematogenous tuberculosis in adults.[1] The perforation of the wall of the air passages was affected by one of two organisms: first, caseation and necrosis of a tuberculous lymph gland which was adherent to the wall of the passageway; second, erosion of the wall of the air passage by the hard and sharp calcified mass in a gland which prior to the calcification had partly eroded the wall and destroyed the elastic fibres. In the first type, a spread of the tuberculous process occurred, while none occurred in the second. The sudden appearance of tubercle bacilli in the pulmonary secretion was considered to be of real diagnostic significance.

Symptomatology, Physical and Radiological Findings: The most consistent findings in tracheobronchial tuberculosis are those related to bronchial obstruction. These findings are as follows: wheeze or rhonchi, particularly unilateral, an area of increased radiability, shift of the mediastinum with elevation of the diaphragm, and opaque lung. These are the findings of obstructive emphysema, atelectasis, or retention of secretion, drowned lung or consolidation. Fifty per cent of patients with these findings show tracheobronchial tuberculosis on bronchoscopic examination.[7]

The following symptoms are reported as occurring in this condition: productive or nonproductive cough out of proportion to the physical or X-ray findings; the cough may be intermittent, intermittently productive or associated with bouts of fever;[6,8] hemoptysis without apparent cause;[6] pain, mild soreness, itching or smothering sensation localized to the lower sternal region;[8] sensation of pressure on the anterior wall of the chest;[13] foreign body sensation in the trachea;[13] and intermittent vomiting.[13]

The following physical findings are reported: wheeze or rhonchi,[7,12] occurring on re-expansion after collapse therapy,[8] and rhonchi elicited by lying on the side with the lower shoulder depressed;[12] recurrent fever;[8,13] anerobic infections in the presence of a lung abscess;[8] and flatness in the interscapular area,[13] and prolonged fever following thoracoplasty.[7]

The following radiological findings have been reported in addition to the ones mentioned above: small areas of density (atelectasis) but without shift of the mediastinum; sudden consolidation of a lobe under collapse; sudden spread of an apparently controlled lesion; blocked cavity with a fluid level; advanced pulmonary tuberculosis with multiple and bilateral cavities;[6] thin-walled cavities or cavities fluctuating in size;[7] hilar flare,[7] although the significance of this is questioned.[8]

BRONCHOSCOPY.

Indications: A suspicion of the presence of tracheobronchial tuberculosis is an indication for the bronchoscopic examination because it is the only means of establishing the diagnosis. Treatment of the lesion may warrant further bronchoscopic procedures directed toward the local treatment of the lesion or for the aspiration and improvement of the drainage of the lung beyond the stenosis. Bacteriological studies of the sputum obtainable only by bronchoscopic aspiration may yield information on the aerobic or anerobic nature of the secondary infection which is significant in the treatment.[10]

Collapse therapy may be profoundly affected by the pres-

ence of tracheobronchial tuberculosis so that an evaluation of the extent and severity of the bronchial lesion and the degree of stenosis which may be present is important. Collapse procedures cause relaxation and shortening of the bronchi, thereby increasing the bronchial obstruction and interfering with bronchial drainage. This is true of phrenic paralysis, artificial pneumothorax and thoracoplasty, although the latter is less apt to be complicated by the presence of endobronchial tuberculosis. Bronchoscopy would, therefore, appear to be indicated for the evaluation of the condition of the bronchi before any type of collapse therapy is instituted.[7]

Contraindications: Bronchoscopic examination is contraindicated in the presence of a hopelessly far advanced tuberculosis in its terminal stage, in the presence of pulmonary hemorrhage, in the presence of an acute upper respiratory infection and in the presence of an ulcerative tuberculous laryngitis. If the tuberculous laryngitis is of the submucosal type or inflammatory type and the patient is cooperative, bronchoscopy may be done if trauma to the larynx is avoided.[7]

Technique: The technique of bronchoscopy in tuberculosis is important because of the sensitivity of the mucosa to mechanical irritation both from the instruments and coughing. The following points are emphasized:[7] premedication consists of three grains of sodium-pentobarbital and one-sixth grain of morphine. The posterior wall of the pharynx is sprayed with 10 per cent cocaine and after a few minutes the pyriform sinuses are sprayed, and then the larynx. The pyriform sinuses are again anesthetized with the cross forceps. One cc. of 2 per cent cocaine is instilled into the trachea, 1 cc. with the patient leaning toward the uninvolved side and 2 cc. while leaning toward the involved side. The 7 mm. full lumen bronchoscope is introduced without the laryngoscope unless there is reason for direct examination of the larynx. The bronchoscope is advanced slowly so that any tracheal or bronchial lesion is not passed. The scope is never passed over such a lesion. The scope is withdrawn slowly and the secretions which have collected around the scope are carefully aspirated. Following the procedure the patient

must be protected from a spread because of the loss of the cough reflex. This is accomplished by placing the patient on his involved side for three hours, by which time the anesthesia will have worn off and the protective reflexes will again be effective.

Bronchoscopic Appearance: The mucosa of the earliest lesions appears granular and reddened. The wall is indurated and edematous so that the cartilaginous rings are less prominent than normal or are not visible. These findings usually occur first about the bronchial orifice leading to the parenchymal lesion. As the lesion spreads, ulceration and granulation tissue appear. As the process continues, the cartilages may be involved and destroyed as is evident by increased mobility of the bronchial walls. In this stage the hyperplastic changes are usually marked. Bronchial ulcers vary in size but they are usually multiple and quite small. The ulcers have irregular edges and their bases are often covered with a grayish exudate. As the healing process starts, activity as well as healing may take place in different areas in the bronchi. The isolated ulcer may heal with very little evidence of scarring, but where there has been more severe involvement of the bronchus more fibrosis and eventually stenosis will develop. The healing process is slow and several months will be required before the granular and friable mucosa and induration disappear.[7]

While a description of the lesion is essential, a classification of the lesions is also helpful:[7]

A. Classification of lesion:

1. Submucosal.

2. Ulcerative.

3. Hyperplastic type: *a.* with ulceration; *b.* without ulceration; *c.* tuberculoma; *d.* destruction of cartilage.

4. Fibrous stenosis: *a.* with ulceration; *b.* without ulceration.

5. Eroding lymph gland.

B. Location and extent of lesion:

C. Associated stenosis (present, absent):

1. Degree of stenosis.

2. Cause of stenosis: *a.* inflammatory edema; *b.* hyperplastic changes; *c.* fibrostenosis.

Bronchoscopic Treatment: Thirty per cent silver nitrate is the most frequent therapeutic solution employed in the treatment of tuberculous ulcerations and granulation tissue.[7,13] It is thought that the degree of stenosis following the healing of the tuberculous process is the result of the disease rather than the therapy.[7] Treatments are repeated every two weeks until all evidence of activity is gone. Patients are then re-examined at increasing intervals for the next six months and then every four or six months. Any recurrence calls for repetition of the treatment. If improvement does not occur after a reasonable time, pulmonary resection is usually advised.[7]

Ultraviolet light and electrocoagulation of the granulation tissue and ulcerations have been recommended,[10] although electrocoagulation did not give as good results as silver nitrate.[13]

Mechanical dilatation of tuberculous stenosis has not given good results.[7,10,13] Galvanic current applied through a copper electrode has seemed to soften the scar tissue.[10] The aspiration of retained secretion is frequently of definite value.[7,10,13]

It is important that the patient with tracheobronchial tuberculosis be given the same general care as the patient with an active parenchymal lesion. This consists of complete bed rest in a sanitarium with dietary and hygienic treatment. In selected cases thoracoplasty may be done and the local treatment of the tracheobronchial lesion continued.[7] While the tracheobronchial lesions generally respond to therapy and improve as the parenchymal lesion improves, this is not always the case. If the bronchial lesion does not respond to therapy and continues to extend, the resulting bronchial obstruction and interference with bronchial drainage eventually cause a high incidence of atelectasis, unexpandable lung,

empyema and anerobic infection.[7,9] Collapse therapy in cases of this sort are limited to thoracoplasty and in many of these the incidence of tension cavities, basal disease and widespread active disease is high.

Pulmonary resection for tuberculosis complicated by tuberculous bronchitis is one of the most recently advocated procedures associated with this disease. Thirty patients with bronchial tuberculosis who have had resection of all or a part of the lung are recorded.[9] Twelve of these patients were considered a desperate risk because they were facing a rapidly fatal course. The mortality rate in this group was 58.3 per cent. The remaining patients were considered a reasonable risk and the mortality rate was 5.5 per cent. Tuberculous bronchitis which must be traversed in doing a resection was not considered a contraindication to the resection. When the individual ligation technique was employed, ulceration in the bronchial stump and contralateral spread were the most frequent complications. It is important to discover those patients with the complication of endobronchial tuberculosis and particularly with ulceration and stenosis so that the dangers of collapse therapy may be recognized and resections performed as indicated before the patients have become desperate risks.

Summary: Tracheobronchial tuberculosis is a complication of pulmonary tuberculosis, the recognition of which is of extreme importance because of its effect upon the prognosis of the disease and the special problems it presents in regard to treatment. In an effort to make the diagnosis, a bronchoscopic examination should be made of all patients who present signs or symptoms of bronchial obstruction, impaired bronchial drainage or bronchial irritation, and the examination should be made prior to the institution of collapse therapy. The bronchoscopic findings vary from a granular indurated mucosa to ulceration and hyperplastic changes with stenosis the end-result. The treatment of choice is the cauterization of the ulcerations and granulation tissue with silver nitrate and the aspiration of secretion from the obstructed bronchi. Pulmonary resection in those cases that fail to respond to

treatment and in whom the disease is progressive offers the patient a chance of recovery.

AEROSOL PENICILLIN INHALATION THERAPY.

The subject of penicillin inhalation in aerosolized form has appeared in the literature during 1944 and 1945.[15 to 23] Although several of the reports are of a preliminary nature, the favorable clinical results warrant a review of the problem. Penicillin therapy is so important a phase of the management of bronchopulmonary disease that while it is not strictly bronchoscopic in character, it is considered here since it has supplanted the bronchoscopic instillation of many of the chemotherapeutic agents recently used by this technique. Its ease of administration and its effectiveness are at once apparent; however, the use of penicillin as well as other agents in this form is not to be considered a panacea as the fundamental surgical principles of establishment and maintenance of adequate drainage must still be applied. Bronchiectasis and lung abscesses require adequate bronchoscopic or surgical drainage now as before the use of these agents. Without adequate drainage in suppurative bronchial or pulmonary disease, lung destruction continues in spite of apparent clinical improvement. The authors point out, too, the fact that clinical improvement is obtained in cases of bronchial neoplasms complicated by bronchial suppuration when penicillin or the sulfa drugs are used. Adequate clinical, radiographic and bronchoscopic examinations should be made prior to the use of penicillin to avoid the error of considering an early bronchogenic carcinoma to be an "atypical pneumonia".

The discussion in this review is limited to aerosol penicillin inhalation therapy. Intratracheal instillations by several techniques have been advocated and should be considered in special instances.[24,25]

Rationale of Penicillin Aerosol Therapy: The difficulties associated with intramuscular and intravenous administration and the possibilities of favorable local action stimulated the search for new means of administration. The administration of certain drugs, principally epinephrine and neo-

synephrin, by means of aerosolization have been of clinical value and in many instances have exceeded the results obtained by parenteral administration.[15] The fact that penicillin is active in high dilutions, its potency is not reduced by organic detritus, and that it does not diffuse readily makes penicillin particularly suited for local therapy.[16]

The process of aerolization with air or pure oxygen does not alter the chemical characteristics nor affect the potency of penicillin.[2,4,14] Particle size is a controlling factor in the penetration into the smaller bronchi of substances in gaseous suspension. Aerosols composed of particles of about 1 micra in diameter are well carried into the respiratory bronchioles.[1,2] Particles of larger size are deposited in the air passages at higher levels.[1]

Technique: The apparatus most commonly used in administering penicillin aerosol consists of cylinder oxygen, a regulator and an aerosol vaporizer with or without economizing features; the penicillin is administered either directly from the vaporizer or by means of an oxygen mask or oropharyngeal oxygen catheter. The penicillin concentration varies from 500 to 100,000 units per cubic centimeter. The solvents are distilled water, physiological salt solution and peppermint water. Individual treatments vary from the administration of 1,000 to 100,000 units.

The two vaporizers commonly used are the DeVilbiss No. 40[2,3,4] and the glass vaponephrin.[1,6] The DeVilbiss nebulizer produces particles with an average radius of 0.54 u. and range of 0.24 u. to 1.18 u.;[2] the vaponephrin produces particles, the majority of which are less than 1 u. in diameter. A slightly larger range of particle size such as are produced by the plastic vaporizers made by the Vaponefrin Company and the Nephron Company may be advantageous.[1]

Various modifications can be made in order to conserve penicillin. A "Y" tube may be inserted into the oxygen tubing which leads to the vaporizer so that the open arm must be covered with the finger before the oxygen will flow through the vaporizer. This conserves penicillin when the aerosol is

inhaled directly from the vaporizer by eliminating the vaporization during expiration. An oxygen mask bag may be inserted into the tubing leading from the vaporizer to the patient so that the aerosol expired and produced during expiration can be made available for the next inspiration; however, the aerosol has a tendency to precipitate on the inside of the bag and consequently this method is not very satisfactory. A glass bulb of 1 liter capacity may be attached to the top of the vaporizer out of the path of the aerosol so that the exhaled and unused mist may collect there and be available for inspiration.[1] The vaporizer may be placed in the mouth and the aerosol inhaled as it leaves the vaporizer. The vaporizer may be connected to an oxygen mask without a valve between the bag and the patient and the aerosol administered in the same manner in which oxygen is administered.[1,3] The vaporizer may be attached to an oropharyngeal oxygen catheter of 10 or 12 French size for administration to infants and smaller children. It is necessary to put a trap between the vaporizer and the catheter so that the larger particles will settle out before reaching the catheter where they may cause obstruction.[1]

Both sodium and calcium penicillin have been used, but the calcium penicillin is preferred because it has less odor and is less irritating to the larynx and tracheobronchial tree.[1] M/50 phosphate buffer adjusted to a pH of 7/ as well as distilled water have been used as solvents,[2] although physiological salt solution is thought to be less irritating to the bronchi because of its isotonicity.[2,6] Equal parts of distilled water and peppermint water have been used in an effort to disguise the odor of the penicillin.[3]

If breathing is continued in a natural fashion during the inhalation of the penicillin, most of the drug will be applied to the bronchi.[1] If inspiration is prolonged to about 15 seconds and the breath held for 15 seconds before expiration and followed by a short rest period, more efficient absorption of the penicillin occurs so that the blood levels and urine excretion are higher.[1,2] This procedure may result in less of the drug being deposited in the bronchi and more in the alve-

oli; however, this may be advantageous even if it is desired to treat a bronchial condition without need of a high blood level. Experience may demonstrate that the best method for maintaining a high blood level is obtained by parenteral administration, while inhalation is best for the local effect in pulmonary and bronchial conditions.[1]

The following is a summary of the most frequently used techniques of the various authors:

Five thousand units per cc. in M/50 phosphate buffer; 25,000 and 50,000 units per inhalation; air flow of 5.5 L.p.m.; with and without face mask with DeVilbiss No. 40.[2]

Two thousand to 100,000, average 40,000 units per cc. in physiological salt solution; 1 cc. per inhalation; 8 L.p.m.; vaponephrin inhalor with glass tube economizer; four to five inhalations a day for five to 10 days.[1]

Eight thousand units per cc. in equal parts of distilled water and peppermint water; 12,000 units per inhalation; oxygen flow of 7 L.p.m.; DeVilbiss No. 40; P. E. M. oxygen mask without the inspiratory valve; 16 inhalations a day for two to seven days.[3]

Five hundred units per cc. in distilled water; 1,000 units per inhalation; oxygen flow of 4 L.p.m.; DeVilbiss No. 40; one inhalation a day for six to 60 days.[4]

Twenty-five thousand or 50,000 units per cc. in physiological salt solution; 1 cc. per treatment; oxygen flow of 4 L.p.m.; vaponephrin inhalor; three to five inhalations daily.[6]

A different method of administration is to vaporize 20 cc. of aqueous penicillin, 10,000 units per cc., in one hour into a room of 3,000 cubic foot size. Exposures of 15 to 30 minutes in this room resulted in excretion of urine which inhibited the growth of penicillin-sensitive staphylococcus aureus in dilutions of from 1:9 to 1:19. The blood of these individuals inhibited the growth of moderately sensitive B. hemolytic streptococcus in dilutions of 1:2 and 1:3. This method may prove to be particularly useful in treating groups of patients and infants and children.[5]

Reactions: Reactions to penicillin aerosol have been few and of a minor nature. Two allergic reactions were observed when inhalations were given within an hour after the injection of a vaccine or pollen immunization.[6] Four cases of urticaria were observed, two of which subsided even though the inhalations were continued,[1] and the other two were allergic patients and were thought to be due to impurities in the drug.[6] Several patients developed a sore throat which subsided within a few days with cessation of the inhalations.[6] Three patients developed fever which subsided when the dosage was reduced one-half for 12 hours and then resumed.[3] Three patients developed slight soreness of the tongue or gums which may have been due to spilling the solution into the mouth; one patient experienced an increase in cough following seven days of treatment, but the cause of this is undetermined; one patient had a slight increase in peribronchial X-ray shadows of questionable significance after one month; two patients developed slight substernal soreness which disappeared even though the penicillin was continued.[1] The highest concentration and dosage given was 100,000 units in 1 cc. of normal salt and no evidence of irritation was noted.[1]

Absorption of Inhaled Penicillin Aerosol: The appearance of penicillin in the urine may be considered proof that the inhaled penicillin was absorbed. Following the inhalation of 25,000 units by means of an oxygen mask, 3.2 per cent was recovered in the urine during the first 12 hours after inhalation. With the nebulizer in the mouth and using the breathing procedure previously described, 60 per cent of the penicillin inhaled was recovered from the urine. This compares favorably with an average recovery of 60 per cent after intravenous injection.[2]

Following the inhalation of 40,000 to 50,000 units at a rate of oxygen flow generally of 8 liters per minute, blood penicillin levels of 0.01 to 0.04 were usually found 15 minutes to one hour after the inhalations. In general, the penicillin blood levels were higher with the larger doses; however, in cases with large amounts of purulent secretion or cases with pulmonary fibrosis, the levels tended to be lower. Using the

special breathing procedure the blood levels were usually higher. Following the inhalation of 100,000 units in 1 cc., 0.4 unit was found in the blood after five to 45 minutes and 0.05 unit after 105 minutes. Administration of 40,000 units to infants and children by means of the oropharyngeal catheter produced blood levels of 0.01 to 0.02 units. Ten to 20 per cent of the penicillin may be recovered from the urine during the first 24 hours after inhalation with the largest amount being excreted in the first hour.[1]

During the daily administration of 192,000 units in 16 inhalations up to 20 per cent was recovered in the urine and most of the blood levels were below 0.03 unit per cubic centimeter of blood. There were unexplained variations in the serum levels.[3]

Effect of Inhaled Penicillin Aerosol on Bacteria in the Sputum: In 15 of 18 patients, sputum cultures taken 24 hours after the termination of treatment failed to show the organism which predominated before the treatment. In one patient, the predominant organism was not sensitive to penicillin, but there was definite improvement in a lung abscess. In eight of the patients the predominating organism was less sensitive to penicillin than the standard organism. In some of the patients the pathogenic organisms recurred later with the reappearance of symptoms.[1]

In a series of 22 patients the inhalation of penicillin aerosol was followed by the rapid disappearance of the Gram-positive bacilli, but the Gram-negative bacilli persisted and in some cases appeared when they had been absent in the cultures made before treatment.[3]

Clinical Results: In a series of 20 patients treated with inhalation of penicillin aerosol, five patients responded very favorably, 10 patients demonstrated moderate improvement, and five patients were unimproved.[1] Four of the patients who responded very favorably had bronchial asthma with bronchial infection; the dyspnea recurred in all and was relieved in two by a second course of treatment. The fifth patient in this group was apparently cured of a pneumonitis and ques-

tionable lung abscess. Of the 10 cases showing moderate improvement, eight had bronchial asthma with bronchial infection and in addition had either pulmonary emphysema or bronchiectasis. Symptoms recurred in four of these patients. Of the remaining two cases in this group, one had a lung abscess which had not shown improvement on intramuscular injections of penicillin and one had bilateral bronchiectasis which was only temporarily improved. Of the five unimproved cases, one had a lung abscess with a closed cavity which did show a decline in fever, two had bronchiectasis with chronic lung abscesses, and two had advanced pulmonary fibrosis.

In a series of 22 patients who were treated with penicillin by the aerosol inhalation method, six patients with bronchiectasis had less cough and sputum than before treatment, but two patients returned to the prepenicillin condition following a respiratory infection. Two patients with questionable bronchiectasis were improved. Of four patients with allergy and some infection, two improved subjectively and two were unimproved. Of five patients with pneumonia, all were improved, although three had an atypical type which had failed to respond to sulfonamide therapy. Two patients with pharyngitis-sinusitis, one of whom also had a lung abscess, recovered. One patient with asthma and bronchiectasis showed no improvement.[3]

Of 40 patients with various respiratory infections, 38 were improved by the inhalation of penicillin aerosol. The best results were obtained in acute and subacute infections. The duration of treatment had to be increased as the duration of the disease increased. Improvement was noted in patients with virus pneumonia who had failed to respond to parenterally administered penicillin.[4]

In a series of over 200 cases with various diseases associated with infections of the respiratory tissues, all were relieved or improved by the inhalation of penicillin aerosol.[6] The conditions included acute and relapsing pneumonitis, sinusitis, sinobronchitis, pharyngitis, intrinsic bacterial asth-

ma, and other manifestations of bacterial allergy associated with upper respiratory infections.

Summary: The inhalation of various dosages of penicillin aerosols of average particle size of less than 1 micra and in concentrations of 500 to 100,000 units per cubic centimeter have been associated with occasional and slight minor reactions or irritations and have caused improvement in the great majority of 282 cases of bronchopulmonary disease associated with infection.

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MISSISSIPPI VALLEY MEDICAL SOCIETY TO MEET IN ST. LOUIS, SEPT. 25, 26, 27, 1946.

The eleventh annual meeting of the Mississippi Valley Medical Society will be held Sept. 25, 26, 27, 1946, at the Hotel Jefferson in St. Louis. Due to the fact that no meeting was held in 1945, all the officers of the society have been retained for another year. These include the president, Dr. Grayson L. Carroll, of St. Louis; president-elect, Dr. Walter A. Sternberg, of Mt. Pleasant, Iowa; first vice-president, Dr. Louis H. Jorstad, of St. Louis; second vice-president, Dr. Elmer E. Nystrom, Peoria, Ill; third vice-president, Dr. E. J. Lessenger, New London, Iowa; secretary-treasurer, Dr. Harold Swanberg, Quincy, Ill.

TEMPORARY DEAFNESS FOLLOWING EXPOSURE TO LOUD TONES AND NOISE.*†‡

H. DAVIS, M.D.; C. T. MORGAN, Ph.D.; J. E. HAWKINS, JR., Ph.D.; R. GALAMBOS, Ph.D., and F. W. SMITH, B.A. (by invitation).

Boston, Mass.

The ears of 15 young men (17 to 21 years) and of four older men (29 to 46 years) were repeatedly exposed at intervals of several days to intense tones of frequencies of 500, 1,000, 2,000 and 4,000 cycles at intensities of 110, 120 and 130 db. for periods of from one to 64 minutes. A noise of continuous frequency spectrum, somewhat resembling airplane noise, was also employed.

Following each exposure, tests were made of the threshold of auditory sensitivity and of the ability to understand words spoken in an A-9 oxygen mask, recorded through an MC-254 carbon microphone heard through a standard headset (Murdock R-14). In many experiments the perception of loudness at various intensity levels was also measured, and several studies were made of the distortion of pitch perception (diplacusis).

Temporary impairment of hearing was regularly produced, but there was no evidence of cumulative injurious effects.

No significant elevation of auditory threshold is produced for tones of frequency lower than the exposure tone. The greatest hearing loss occurs at a frequency about half an octave above the exposure tone. With brief exposures the loss may be confined to the two octaves above, but with the longer exposures the hearing loss may be quite extensive for all tones above the exposure frequency.

*Read at the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Inc., Boston, Mass, Jan. 9, 1946.

†The present article is the summary of a 70 page report of the Committee on Medical Research dated Sept. 30, 1943.

‡The work described in this paper was done under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and Harvard University.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 11, 1946.

Taking as a measure of hearing loss the average loss throughout the two octaves above the exposure tone, we find that:

1. One thousand cycles and 2,000 cycles are about equally effective in producing hearing loss.

2. Four thousand cycles is much more effective and 500 cycles is much less effective than 1,000 or 2,000 cycles.

3. Hearing loss develops most rapidly during the first minutes of exposure and then more and more slowly.

4. More intense tones usually cause greater hearing losses, but one-minute exposures to 2,000 cycles may be *less* effective at 135 db. than at 125 or 130 db.

5. The complete relations of hearing loss to frequency, intensity and duration are complex, and the graphs of Section II (of the report of the Committee on Medical Research dated September 30, 1943) must be consulted for details.

6. Recovery of hearing usually begins rapidly and then progresses more and more slowly. Recovery from a 60 db. hearing loss may require four or five days to be complete. Recovery tends to be slowest for frequencies of about 4,000 cycles, regardless of the frequency of the original exposure tone.

Some men are much more susceptible than others to the production of hearing loss. There are differences also as to the part of the frequency range most readily affected, and also in the rate of recovery from a given degree of loss. Occasionally any one man may deviate considerably from his usual susceptibility. The results of exposure to 2,000 cycles are more variable than for the other frequencies.

The impairment of hearing produced by exposure to loud tones or noise is of the "variable" or "nerve deafness" type. In spite of elevations of threshold of 50 or 60 db., there may be little or no loudness loss for sounds at the 100 db. loudness level. Losses at this level rarely exceed 6 db. *The audiogram alone is not an adequate measure of the impairment of auditory function.*

The modification of loudness perception is shown to be more complex and variable than has hitherto been described either for "nerve deafness" or for masking with a background noise of continuous spectrum.

Exposure to a pure tone that causes a hearing loss that is restricted to a relatively narrow range of frequencies may cause very severe distortion of pitch perception (diplacusis). Tones of certain frequencies sound noisy and impure or they may be abnormally elevated in pitch by as much as three-quarters of an octave. The major displacements of pitch are always upward. Exposure to a band spectrum noise (like airplane noise), which causes a widespread hearing loss that is usually most severe in the high frequency range, is relatively ineffective in producing diplacusis.

The impairment of understanding of speech is more closely related to the *overall loudness loss at the intensity level at which the speech is heard* than to the threshold audiogram or to the loudness loss in any special portion of the speech frequency range (400 to 4,000 cycles). Prolonged exposure to an intense 500-cycle tone or to noise of wide frequency spectrum causes severe articulation loss at a low (40 db.) loudness level but only moderate loss at a high (100 db.) level. The articulation loss for loud speech following the 500-cycle exposure tends to be the greater of the two even though the average hearing loss measured by audiogram is less, probably because of the greater diplacusis produced by the exposure to the pure tone. Exposure to an intense 1,000-cycle tone may or may not produce a measurable articulation loss for loud speech, and exposures to 2,000 and 4,000 cycle tones cause but little articulation loss, even at the 40 db. loudness level, for speech heard through a standard Army headset.

MANAGEMENT OF FRACTURES INTO THE NASAL SINUSES.*

JOHN J. SHEA, M.D.,
Memphis, Tenn.

The maxillary is the most frequently entered sinus during a compound facial injury. The management of such an accident can be simple or very complicated according to the approach. After a careful study of the occlusion of the teeth, the relative position of the eyes and the general appearance of the symmetry of the face, an experienced surgeon can anticipate the Roentgenological findings. Unfortunately, an ordinary Roentgenological study does not reveal the true extent of the fractures and only after stereoscopic or planographic studies can the fractures be appreciated.

INSPECTION.

The inspection of a "compound facial injury" patient instantly reveals a unilaterally swollen face if the fracture is limited to one side, or a "horse face" if the fracture is bilateral. The latter is an elongated face due to a dropping of the upper teeth with their alveolar attachment. The degree of limitation of the movement of the lower jaw depends upon whether the zygomatic arch interferes with the free excursion of the coronoid process of the inferior maxilla or if there is sufficient clearance. Palpation of the rim of the orbit will ascertain if the fracture extends into the orbit, and the usual displacement is along anatomical lines due to the orbital articulation.

ROENTGENOLOGICAL STUDY.

As many views as necessary should be made to study the extent of the fractures; some with the mouth open and others with it closed to visualize the relative position of the coronoid

*Read at the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc., Richmond, Va., Jan. 7, 1946.

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process and the zygomatic arch. Stereographic studies are better than flat plates, and laminagrams will visualize fractures not revealed by either of the preceding studies.

WHEN TO OPERATE.

If the patient has not suffered an intracranial injury, the sooner the fractures are reduced the better are the results;

Fig. 1a. Illustration of balloon within the antrum for support of orbital floor.

but if there is any question of an intracranial insult it is best to wait. If during this delay an infection develops, it is necessary that chemotherapy be instituted and that the infection subsides before any manipulation of the parts be undertaken. The daily administration intravenously of calcium gluconate 10 cc., buffered by paratyphoid extract 1cc., subcutaneously will relieve the swelling of the soft parts.

Wounds of the soft tissue should be thoroughly cleansed and repaired with the skill of a plastic surgeon. Should there

be any question of an intracranial complication, an immediate consultation should be held with the neurological department.

OPERATION.

The majority of fractures involving the maxillary sinuses may be handled by a very simple routine and with a minimum number of instruments. The anesthetic of choice is pentothal sodium with the usual local anesthesia of a radical antrum operation. An incision is made as for a Caldwell-Luc operation, and the anterior wall of the antrum exposed. If the zygomatic arch is depressed, the arch can be elevated through this approach by three manipulations. The elevator of choice requires but one quality and that is stability. The instrument is first inserted under the malar bone and, using the temporal region as a fulcrum, displaced forward. Second, the instrument is adjusted under the center of the arch and the arch is sprung outwardly. The last manipulation assures a clearance for the coronoid process. If the line of fracture bisects the anterior wall of the antrum, the segments are separated and retracted sufficiently to allow inspection of the interior of the maxillary sinus. Frequently the floor of the orbit is fractured and depressed without rupturing the eyeball; likewise, the fracture may extend around the base of the malar process and the contents of the pterygoid maxillary space be pushed within the cavity of the antrum. The antrum is now packed with gauze as tightly as possible. This manipulation is the safest means of elevating the roof of the antrum, which is the floor of the orbit. A nasoantral window is made and a Ferris Smith antral balloon* is inserted and inflated either with air or water to maintain the fragments in their proper position. The wound is closed with interrupted sutures and the nose gently packed to control hemorrhage. This packing is removed in 24 hours, but the balloon may remain *in situ* from five to seven days. The operation will consume from 15 to 30 minutes and obtain a uniformly good result without the necessity of wiring or the use of complicated external appliances.

*These balloons may be secured from C. R. Bard, Inc., 79 Madison Ave., New York 16, N. Y.

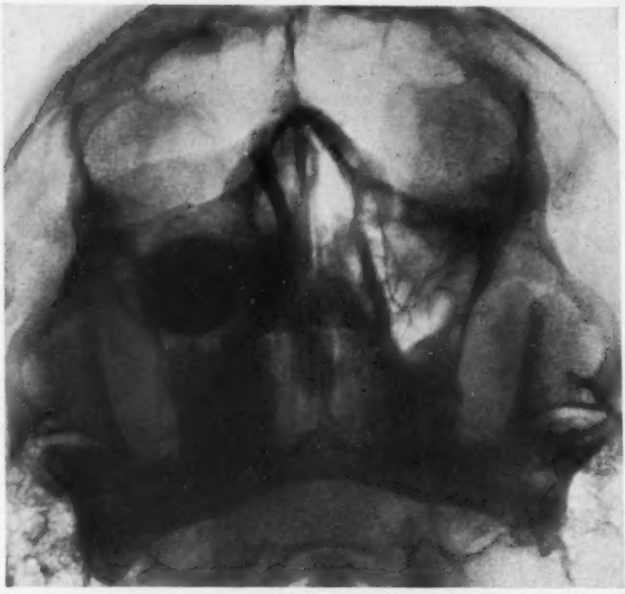

Fig. 1b. Illustration of balloon within the antrum for support of orbital floor.

This operation can be performed through an enlarged naso-antral window especially in young adults, when there is no depression of the zygomatic arch, thus avoiding section of the nerves to the teeth.

CONCLUSIONS.

A simple procedure similar to steps followed in a Caldwell-Luc operation is sufficient for the management of the repair of fractures in the ordinary "compound facial injury."

1018 Madison Avenue.

THE VALUE OF INDIVIDUAL HEARING AIDS FOR HARD OF HEARING CHILDREN IN PUBLIC SCHOOLS. REPORT TO THE SUB-COMMITTEE OF THE COMMITTEE ON PROBLEMS OF DEAFNESS OF THE NATIONAL RESEARCH COUNCIL.*

THE OTOLOGICAL EXAMINATION AND FOLLOW UP.

EDMUND PRINCE FOWLER, M.D.,
New York, N. Y.

The primary purpose for examining these children otologically was to match them in pairs according to similarities in degree and type of hearing loss; also these pairs were to be matched as near as possible psychologically, behaviorly, etc. Over 250 children were examined by me and 100 of these were chosen for pairing. Fifty-two children were paired (26 pairs). The first child was provided with a hearing aid and the other was not. The 48 remaining children were not closely pairable, but half of them were supplied with hearing aids. One extra child was used as a spare.

All of these children had been tested at least once previously by both 4-A and 2-A audiometers in the public schools. My tests were made in an efficiently soundproofed room in my office; the examinations and tests were repeated at least once a year, over a period of three years (12 children had only two tests).

It is customary to estimate gains and losses in hearing by decibels (usually averaging the change), but this may be very misleading, because each frequency rates a different importance for hearing the speech sounds. To obtain a truer index I have chosen to use the percentage of capacity for hearing speech (instead of threshold decibel losses). By so doing we apply a measure which corresponds more closely with clinical experience.†

*Under the direction of Rudolph Pintner, Ph.D., and Arthur I. Gates, Ph.D.

†Fowler's Method of weighting has been adopted by the Consultants on Audiometers and Hearing Aids of the American Medical Association. (No allowance was made for the recruitment phenomenon or variable ratio between the two ears.)

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The scatter chart (see Fig. 1) shows the distribution of the losses along the longitudinal base line, according to the original percentage of loss, at the point of bisection of the ordinates, on which in each case the subsequent gain or loss

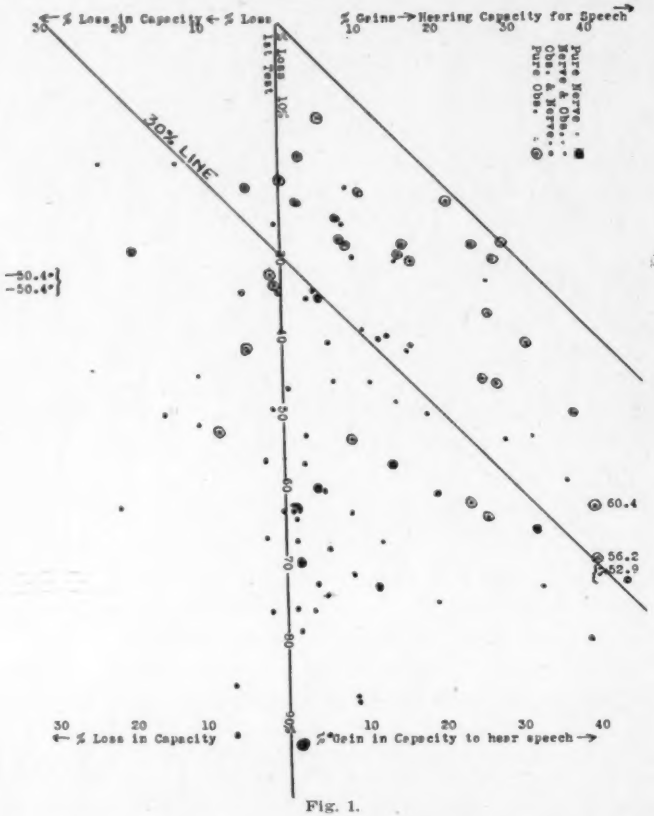


Fig. 1.

was plotted. In each instance this shows the capacity to hear speech, not necessarily the proficiency in understanding speech. The soft voice was used for the latter purpose.

Diagnostically the cases fell into four main groups indicated on the chart by the symbols shown opposite each group:

Pure nerve deafness	8	● large dot
Nerve deafness with some obstructive deafness	57	• small dot
Obstructive deafness with some nerve deafness	5	○ small circle
Pure obstructive deafness	31	⊙ 2 concentric circles

Under pure obstructive deafness there is included all cases of dry and all purulent otitis media, and mastoiditis, and one case diagnosed tentatively as otosclerosis. In the combined nerve and obstructive deafness cases the lesion causing the more severe loss is placed first, *i.e.*, "nerve and obstructive," or "obstructive and nerve," as the case may be.

The percentages of loss in capacity to hear speech were obtained by using Fowler's Method.

As was to be expected, the most spectacular gains in hearing were obtained in the pure obstructive cases, having no more than a 30 per cent loss at the first testing. Of 15 such cases all but three showed improvement at subsequent tests.

It will be noted (see Table 1) that one-third of the children had no hypertrophied pharyngeal lymphoid tissue. Of those showing hypertrophy about one-half remained unchanged and about one-half showed definite diminution.

Fifty-four of the children had hypertrophied turbinates (54 per cent), and 15 per cent of these showed distinct improvement when last seen.

In 8 per cent of the children, tonsil and adenoid operations were recommended, and in 6 per cent radon treatment was advised to diminish the lymphoid tissue near the Eustachian tubes. In only one instance was the latter carried out.

Fifty of the children were recommended for treatment other than otolaryngological.

Whereas in each instance the parents of these children were instructed in guidance and care, no medicinal measures were prescribed except antiseptic and cleansing eardrops for the suppurative ears, precautions in swimming, and guidance in the prevention of colds; however, it cannot be denied that the mere fact of consulting a doctor and being made to realize the importance of the hearing disability, gained for many of

TABLE 1.

No.	Type of Deafness*	Rt.	Number with Tinnitus Lf.	Both	Norm.	Lymphoid tissue in nasopharynx Hyp.	No chg.	Dim.	Changes in the Hearing: No chg.	Actual with gain	with loss	Less than 10% gain or 5% loss —no chg.	Impvd. 10% or more —gain	Lost 5% or more —loss
8	Pure Nerve	—	—	5	1	7	3	4	5	3	0	62.5%	37.5%	0.0%
57	Nerve & Obstr.	7	2	31	14	43	20	23	29	18	10	58.7%	31.5%	9.8%
5	Obstr. & Nerve	1	—	1	3	2	1	1	1	4	0	20.0%	80.0%	0.0%
31	Pure Obstr.	4	4	18	10	21	11	10	12	17	2	38.7%	54.7%	6.5%
		12	6	55	28	73	35	38	47	42	12			

*In only one instance were the lesions bilaterally etiologically dissimilar. (One ear was totally deaf.) This case was included under "Pure Obstr." because the total deafness antedated the primary test and the change in hearing was due wholly to the change in the obstructive deafness in the hearing ear.

these children better care and treatment than they had had in the past. No vitamins with the exception of vitamin D in winter were prescribed.

Dr. Irving Lorge reports:

"It is significant that the variation in hearing among individuals is greater than the variation between the means of the aid and the no-aid groups respectively, but the variation among individuals is so great that it obscures any genuine differences between groups.

"The variation in the gains of individuals is so variable that any distinction between aid and no aid cannot be clearly revealed with the number of cases utilized in this experiment." In other words, use of the hearing aid did not have any effect on the basic hearing capacity.

None of the eight *pure nerve deafness* cases showed a further loss during the three years of observation. One showed a spectacular gain of 33 per cent (to within 2 per cent of the 30 per cent line). Such gains are not unusual in children.

The gains in the *nerve-obstructive* cases were spectacular in some instances, and considerable in the great majority of cases, but only 11 per cent of these nerve-obstructive deafness children regained enough hearing to function well for a soft voice more than five feet away (less than a 30 per cent loss). Nevertheless, any gain in hearing, no matter how slight, in nerve cases contributes more than meets the eye in the threshold change in capacity.

The five *obstructive-nerve* cases showed changes as follows:

1 from 72.0 to 26.6 a gain of 45.4%
1 from 79.6 to 40.4 a gain of 39.2%
1 from 60.3 to 41.1 a gain of 19.2%
1 from 41.5 to 24.9 a gain of 16.6%
1 from 34.3 to 35.2 a loss of 0.9%

Sixteen of the 31 *pure obstructive* cases showed a hearing loss at first test of more than 30 per cent. In four of these there was subsequently a greater loss, only one being more than 5 per cent. Of those that gained, all but three improved

to the 30 per cent line or above. Only one lost enough to bring it more than 8 per cent below the 30 per cent line.

Fifteen of the 31 *pure obstructive* cases showed at the first examination losses not over 30 per cent. At the last test one of these showed no loss or gain; one showed a loss of 19 per cent; one a loss of 4.5 per cent; 12 showed gains which, with two exceptions, sufficed to bring them over the 25 per cent line.

From the scatter graph it is apparent then that the changes (including all losses or gains) indicate that 39 of the children now have no greater than a 30 per cent loss, and that 15 others showed a loss of less than 40 per cent. At the first test these figures were 20 and 15 children respectively. In other words, more than half of all the children could at the last test hear the moderately loud voice quite well at distances of less than five feet, whereas at the first test only one-third of the children could hear this well.

The data which stand out most sharply are: *1.* the number of children who improved to some extent (namely, 78 per cent) compared to the number who lost to some extent (namely, 22 per cent); *2.* the number who improved over 20 per cent (namely, 22 per cent); *3.* the number who at the last test showed less than a 30 per cent loss (namely, 39 per cent); and *4.* the number who were deafened (to some extent at least) from auditory neural lesions (70 per cent).

The distance at which the soft voice was heard varied directly with the per cent gain or loss of capacity to hear the speech frequencies. I feel certain that more frequent examinations of the children would have resulted in higher percentages of gain.

The data definitely show that over 75 per cent improved to some extent, and at least one-third of the children showed a marked restoration of hearing. About 40 per cent improved to the 30 per cent loss level.* Had some special "cure for deafness" been employed, these statistics might be used to

*A 30 per cent loss is equivalent to about a 36 weighted db. loss in both ears (Fowler's weighting). Also to a 70 per cent loss in one ear and no loss in the opposite ear. All percentage losses are overall losses.

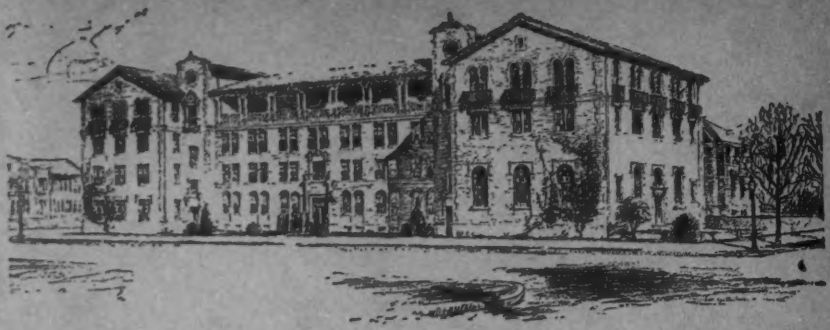
show that the "cure" was responsible for the remarkable "restorations" of hearing. Obviously any such claim would be unwarranted.

SUMMARY OF DR. MILDRED B. STANTON'S REPORT.

The effect from wearing the hearing aid upon school achievement and personality was determined by the Stanford Achievement Test, Aspects of Personality and Pupil Portrait —"there was great variation in the time the children wore their aids — from 100 hours to over 700 hours. There was remarkably little breakage." Statistically there were no reliable differences between the experimental and other groups with respect to the achievement or personality inventories. There was, however, a trend toward improvement in social and school adjustment, and achievement. The parents, teachers and investigators frequently remarked that "many of the children wearing hearing aids seem happier and less tense." The most difficult hazard was the overcoming of distaste, or the fear that wearing a hearing aid would set up an abnormal social contact status or interfere with popularity or attractiveness to others. It was not only the children who voiced these apprehensions, but also the parents, some of whom thought that wearing the aid would make their daughters less marriageable.

It was apparent that to remove these hazards the hard of hearing child should begin wearing the hearing aid on starting school. This might also prevent the habit of "school failure patterns" and would aid in lip-reading and speech correction. It was felt that more emphasis should be put upon teaching the children how to use the aid to the best advantage. To obtain full benefit from their aids, a careful training program for the teachers of regular classes should be developed. Too many teachers harbor the notion that if the child would just listen he could hear. Tensions in the home of many of the hard of hearing children could not be resolved without first removing some of the "unhappy behavior patterns." No single procedure will solve all the difficulties of the hard of hearing children.

140 East 54th Street.



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CONTENTS